The
VETERINARY RECORD
Founded by William Hunting, F.R.C.V.S., in 1888

7, MANSFIELD STREET, PORTLAND PLACE, LONDON, W.1. MUSEUM 6541

No. 13 MARCH 26th, 1955 Vol. 67.

Registered for Transmission as a Newspaper *Price: 2s. post free*

Contents

General Articles :

A Contribution to the Epidemiology of Parasitic Bronchitis in Calves 229
J. F. Michel, M.A., DIP. AGRIC. SCI.
J. W. Parfitt

Hexamitiasis of Turkeys ... 236
J. E. Wilson, B.SC., M.R.C.V.S., F.R.S.E.
D. Slavin, M.R.C.V.S., DR. VET. MED. (LEIPZIG)

Current Literature :

Abstracts 242
Book Review 243

News and Comment 245

Correspondence :

Pregnancy Toxaemia 247
J. A. Toovey, B.SC., M.R.C.V.S.

The Diseases of Sheep 248
G. A. M. Sharman, B.SC., M.R.C.V.S.
T. Graham Marr, B.SC., M.R.C.V.S.

Metaldehyde Poisoning 248
Olwen D. James, M.R.C.V.S.

A Contribution to the Epidemiology of Parasitic Bronchitis in Calves

BY

J. F. MICHEL and J. W. PARFITT
Ministry of Agriculture, Veterinary Laboratory, Weybridge

Introduction

EPIDEMIOLOGICAL studies on *Dictyocaulus viviparus* have in the past been hampered by the lack of a technique for the recovery of larvae from herbage samples. Methods normally used for the enumeration of trichostrongylid larvae proved ineffective owing to the inactivity of those of *D. viviparus*.

The investigation here reported began when two infected calves were placed in a half acre paddock close to the laboratory in order that they might be observed. Subsequently the paddock was used for the elaboration of a technique for enumerating lungworm larvae on herbage. After a short time this began to be determined regularly and other animals were put in and taken out in order to observe the effect upon them and the effect that they in turn had on the level of infestation on the herbage.

The results of what might perhaps be regarded as preliminary observations are presented at this stage not only on account of the tentative conclusions that may be drawn but also because they illustrate the possibilities of the development of this kind of investigation.

Materials and Methods

The half acre paddock used in these investigations was a moderate Agrostis-Rye grass pasture with only a small content of wild white clover. The calves used were of a variety of breeds, but were all reared in loose boxes under conditions that had been found to prevent accidental infection with lungworms. With the exception of the first two calves, Nos. 1891 and 2064, all the calves put into the paddock were free from infection.

The following technique was used for the estimation of *D. viviparus* larvae in faeces. Ten grammes of faeces collected from the rectum are shaken up with approximately 70 c.c. of water. The resulting suspension is poured into a screen, eight inches in diameter, having 80 meshes to the linear inch, through which it is forced with the aid of a strong jet of water. The fluid passing through the screen (which should total approximately 500 c.c.) is caught in a bowl from which it is poured into a round-bottomed pint measure. It is allowed to sediment for at least three hours, after which time the larvae will have settled out. The supernatant is syphoned off to leave approximately 60 c.c. of sedi-

ment for at least three hours, after which time the larvae will have settled out. The supernatant is syphoned off to leave approximately 60 c.c. of sediment which is transferred to four Clayton Lane centrifuge tubes. After centrifugalisation the supernatant is replaced by saturated brine, cover glasses placed on the tubes and a standard salt flotation carried out. The larvae are then washed off the cover glass with water and centrifugalised. The sediment is examined under the 2/3 inch objective and each larva seen is counted as representing one per 10 gramme faeces.

The concentration of the larvae of *D. viviparus* on the herbage was determined by the following method. Grass is collected from 800 points on the area to be sampled which are selected in the manner of Taylor's (1939) method. Two workers follow predetermined W-shaped routes across the paddock, stopping 100 times and cutting small pinches of grass close to the ground from four places at each stop. The quantity cut from each place is adjusted so that each collector gathers approximately 8 oz. in all.

These two samples are weighed and soaked in water in two-gallon buckets. After at least two hours the grass is agitated and then removed in small handfuls which are well squeezed and transferred to a second bucket of water in which the process of washing is repeated, after which the grass is discarded.

The buckets are left overnight for sedimentation to take place, the supernatant in syphoned off to leave approximately 500 c.c. in each, which, after being passed through a screen having 80 meshes to the linear inch, is transferred to a round-bottomed pint measure.

From this point the procedure is identical with that described above for the enumeration of larvae in faeces.

The two washings of each 8 oz. sample are handled and counted separately and subsequently added together.

It is impossible to determine the percentage recovery of larvae obtained by this method but by adding larvae to samples of clean grass and at various stages in the procedure some indication of the losses can be obtained. Thus some 18.9 per cent. are lost in the process of washing the larvae off the grass, 10.2 per cent. in sedimenting in the bucket, 21.3 per cent. in sedimenting in the pint measure, and 25 per cent. in the process of flotation. The average recovery of larvae artificially placed on grass was 43 per cent.

The larvae recovered by this technique from herbage samples taken during the course of this investigation were always in the third stage of development.

Figs. 5 and 6 represent typical microscope fields showing larvae obtained from herbage by the procedure that has been described. The coiled larva seen in the centre of Fig. 5 is one which would be termed "clear" while that in Fig. 6 is "granular." It is our experience that the appearance of a larva gives a good indication of its age. Thus a granular larva is up to two weeks old, one only slightly granular is between one and three weeks old, while a clear larva is more than three weeks old.

Although this technique is of considerable value, a more sensitive one would be even more useful, experience having shown that counts of less than one larva per pound of herbage are of significance in the production of disease.

Course of the Infection on the Paddock and in the Calves

The observations made are summarised in Figs. 1, 2, 3, and 4 which represent the fluctuation in the concentration of larvae on the herbage and in the number of larvae in the faeces of the calves.

The heavy black line represents the level of infestation of the herbage measured in larvae per pound of grass. Above this are other graphs drawn in a lighter line representing the count of larvae per gramme of faeces of the animals grazing in the paddock. Details of the animals used are summarised in Table I.

The earliest observations are shown on Fig. 1. The two infected calves, Nos. 1891 and 2064, which had been put in the paddock so that they might be observed, had been there for some days when their faeces were first examined on October 3rd. The first reliable grass count was done some three weeks later. The way in which the larvae passed by these two calves gave rise to a high peak in the grass count can clearly be seen. This was followed by a relatively rapid decrease.

On November 9th a small part of the paddock was fenced off with a double fence and designated "D." The fall in the grass count on this piece was parallel with that in the rest of the paddock. On May 17th, 1952 when paddock "D" had been free of cattle for five and a half months, a young calf, 2139, was put in. It was slaughtered on June 24th, but no lungworms could be recovered from it. Apparently no larvae had survived.

When two strong calves, 2070 and 1910, were put in the main portion of the paddock on December 4th they acquired considerable infections. Though large numbers of larvae were now being passed on to the ground the response of the grass count was very slight. It is true that there was a slight rise in the second half of January and the first half of February, but it would appear that climatic or other conditions in January and February were not favourable to the translation of faecal larvae into larvae on the herbage. Calves 1910 and 2070 were removed from the paddock on January 30th.

Two yearlings, 1926 and 1932, were introduced into the paddock on January 8th and removed on March 14th. These became infected only to a very slight extent partly, perhaps, on account of their possessing a measure of age resistance but chiefly because of the unfavourable conditions for the transmission of infection apparently obtaining in winter.

Two further calves, 2133 and 2131, were introduced on March 11th. There appears to have been some difference in their individual susceptibility but this difference was far more marked in faecal larval

TABLE I.

No.	Paddock	Age in months	Date put in paddock	Removed, killed or died	Time in paddock days	Lungworms recovered *post mortem* >3cm.	>2cm.	>1cm.	>3mm.	<3mm.	Remarks
1891	Whole	6½	30.6.51	D. 7.10.51	99	2,400	143	—	—	—	
2064	Whole	2½	9.9.51	D. 31.10.51	52	1,810	724	236	—	—	
1910	Whole	10½	4.12.51	R. 30.1.52	57	718	199	43	23	16	Killed 22.2.52
2070	Whole	4½	4.12.51	R. 30.1.52	57	470	28	15	23	230	,, 9.2.52
1932	Whole	10	8.1.52	K. 14.3.52	66	—	—	—	12	50	
1926	Whole	11	8.1.52	K. 13.3.52	65	—	—	—	6	20	
2133	Whole	3½	11.3.52	R. 17.5.52	67	—	—	—	—	—	
2131	Whole	4	11.3.52	R. 17.5.52	67	—	—	—	—	—	
2138	B_1 & B_2	5	17.5.52	D. 10.6.52	24	4,035	6,410	3,589	2,228	—	
2157	B_1 & B_2	4	17.5.52	D. 7.6.52	21	9,155	13,028	16,420	—	—	
2311	B_1	4	31.7.52	D. 19.9.52	50	70	114	26	35	—	Phenothiazine poisoning
2312	B_2	4	31.7.52	D. 27.11.52	119	80	95	95	189	136	Parasitic gastritis
2313	B_2	4	16.9.52	D. 29.10.52	43	1,677	680	124	162	33	
2328	B_2	7½	6.2.53	D. 19.6.53	132	2,830	10	26	207	1,381	
2307	B_2 & C	9½	4.5.53	K. 30.7.53	86	—	—	—	—	—	
2466	B_2 & C	5½	25.5.53	D. 23.6.53	29	5,340	1,282	15,211	13,097	75	
2160	C	5	16.6.52	D. 6.10.52	112	562	1,062	5,940	1,318	—	Parasitic gastritis
2251	C	2½	16.6.52	D. 22.9.52	98	89	29	6	3	3	Phenothiazine poisoning
2341	C	5	6.2.53	D. 11.5.53	94	255	1,062	594	1,338	—	Parasitic gastritis
2233	A	4	7.7.52	D. 20.9.52	75	1,744	2,690	1,178	501	640	Death precipated by Phenothiazine poisoning
2139	D	5	17.5.52	K. 24.6.52	38	—	—	—	—	—	
2265	Hay from A.	2	4.7.52	K. 18.8.52	45	—	—	—	—	—	

counts than in the mild symptoms that they showed. 2133 passed fair numbers of larvae and this was reflected in a sharp rise in grass count. On May 17th, 2133 and 2131 were taken out of the paddock, which was now divided into four. These four enclosures were designated A, B_1, B_2, and C. B_1 and B_2 were left confluent and two calves, 2138 and 2157, put into them. Both died within a very short time of acute parasitic bronchitis on June 10th and 7th respectively. This must have been the consequence of the high level of infestation on the ground when they were introduced. In neither did the infection have time to become patent and so these calves passed no larvae on to the paddock. On June 11th

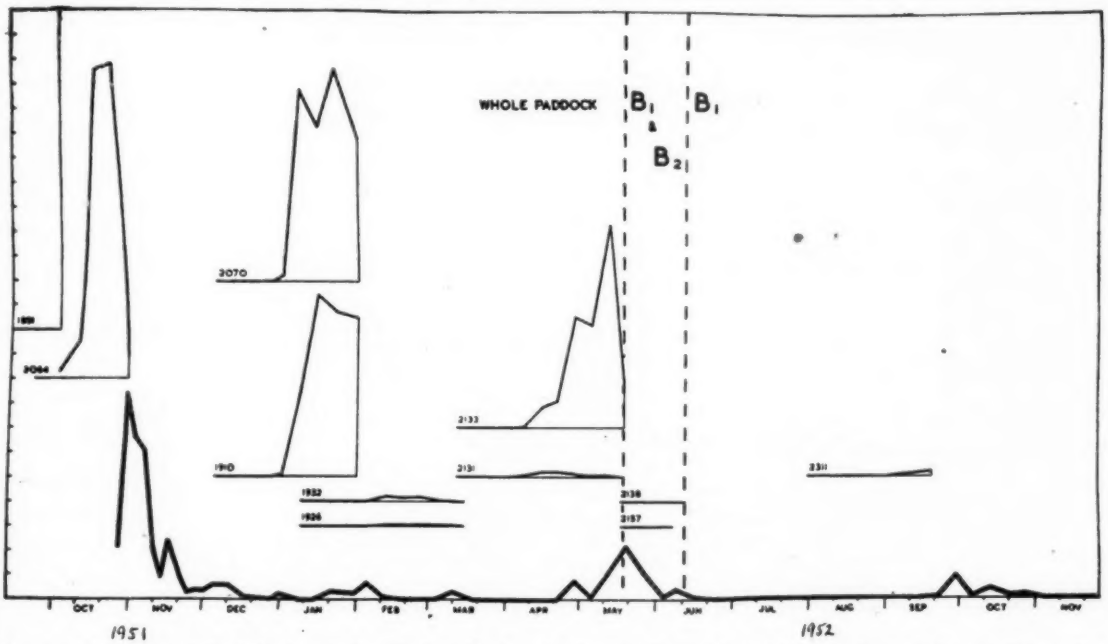

FIG. 1.—The course of the infection on the whole paddock and subsequently on B_1 and in the calves grazing on them. One division on the vertical scale represents 20 larvae per lb. of herbage or 50 larvae per gramme of faeces.

"B_1" was separated from "B_2." Fig. 1 follows the happenings on "B_1," which was allowed to lie empty until July 31st when a calf, No. 2311, was put in. This acquired only a light infection and passed but very few larvae. None the less an appreciable infestation on the herbage resulted. It may be that the great quantity of herbage in B_1 throughout the time that 2311 was on it, together with the fact that this calf scoured violently, assisted in making the larvae passed in the faeces available on the herbage.

By this time parasitic gastro-enteritis had begun to be a problem and had already resulted in a number of deaths on other plots. It was therefore decided on September 17th to institute phenothiazine therapy although the poor condition of the animals meant that this was attended with some risk. 2311 died on September 19th, apparently from phenothiazine poisoning.

Fig. 2 represents the happenings in "B_2," but for the sake of continuity the last part of the record of the whole paddock and of "B_1 and B_2" is also shown. On July 31st a calf, 2312, was introduced and six weeks later on September 16th a second calf, 2313. The first calf acquired a light infection but ultimately died from parasitic gastro-enteritis on November 27th. The larvae which it passed must have raised the herbage infestation so that the second calf acquired a much greater lungworm infection and died on October 29th, apparently of parasitic bronchitis. The grass count did not rise appreciably until the end of October, and thereafter fell away very gradually, positive counts being obtained until mid-February of 1953.

A further calf, No. 2328 was put in on February 6th and this animal acquired only a light infestation in the first instance. On May 2nd, 1953 the fence dividing "B_2" and "C" was removed. Herbage samples continued to be taken separately from the two enclosures, and results are recorded on Figs. 2 and 3. For convenience of description it will be better to deal next with "C" and then to proceed to a consideration of events taking place after the removal of the fence dividing "B_2" and "C."

Happenings on enclosure "C" are shown in Fig. 3. Two calves, Nos. 2160 and 2251, were put in on June 16th, 1952 and in the first instance acquired only moderate lungworm infections. 2251 died on September 22nd from phenothiazine poisoning, 2160 on October 6th apparently from parasitic gastro-enteritis, though its appreciable lungworm burden was probably a contributory cause of death.

The effect of the larvae passed by these calves was a rise in the grass count which fell away gradually, the last positive sample being obained in January 1953. When calf 2341 was put in on February 6th it acquired only a small infection of lungworms but was removed from "C" on April 28th and died from parasitic gastro-enteritis on May 11th. The small number of larvae passed in its faeces resulted in an appreciable grass count.

"C" was amalgamated with "B_2" on May 2nd, 1953. As has already been seen 2328 had become lightly infected on "B_2" and it appears that the larvae it passed and which had raised the grass count, augmented the infection it was carrying as may be seen from the rise in its faecal larval count that took place during the first three weeks of May.

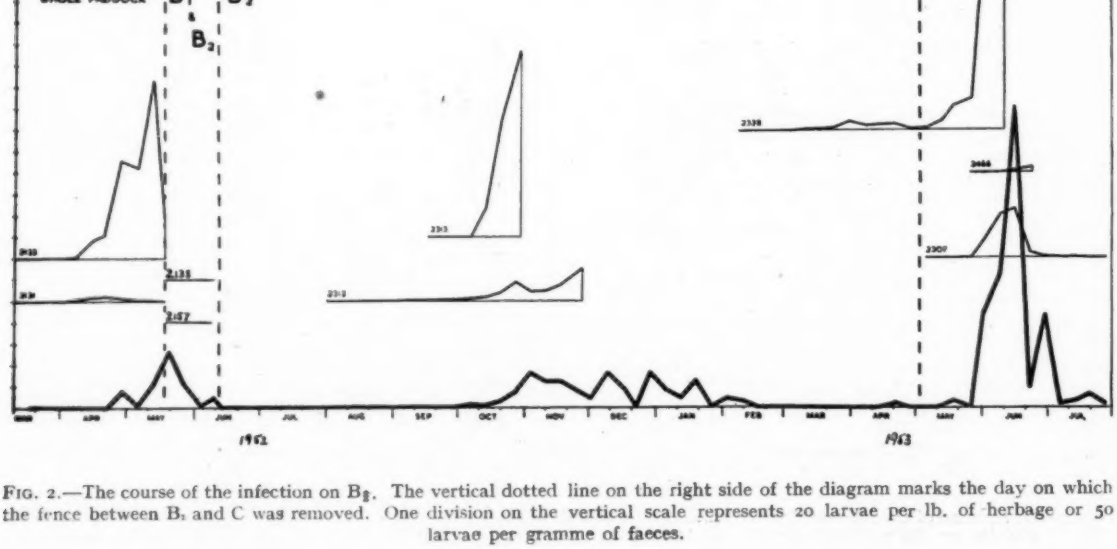

FIG. 2.—The course of the infection on B_2. The vertical dotted line on the right side of the diagram marks the day on which the fence between B_2 and C was removed. One division on the vertical scale represents 20 larvae per lb. of herbage or 50 larvae per gramme of faeces.

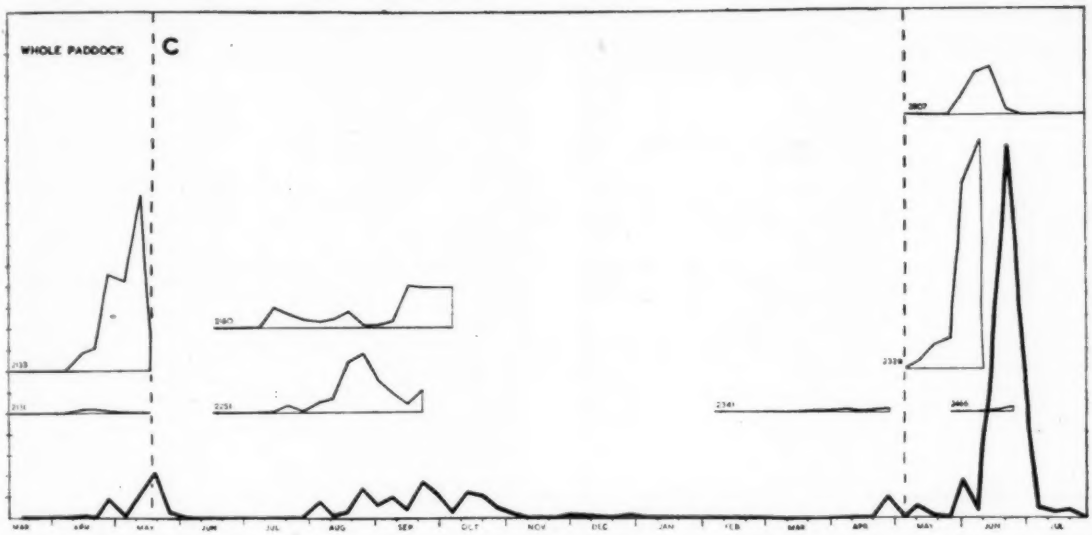

FIG. 3.—The course of the infection on C. The vertical dotted line on the right side of the diagram marks the day on which the fence between B_2 and C was removed. One division on the vertical scale represents 20 larvae per lb. of herbage or 50 larvae per gramme of faeces.

The grass count in "C" was even higher and the effect of this when 2328 came to be exposed to it was reflected in the great rise in the faecal larval count that took place at the end of May. This in turn had a most spectacular effect on the grass count on both "B_2" and "C." The count on "B_2" reached a peak of 280 larvae per pound of herbage and that on "C" a peak of 350. This difference cannot be considered to be of any material significance.

On May 4th a nine-and-a-half-month-old calf, 2307, was put on "B_2 and C." This animal had been out for a single day on paddock "B_2" on December 18th and had acquired an exceedingly light infection, which remained patent for 10 days, the faecal larval count being never more than one larva per gramme.

The effect upon it of exposing it to infection on "B_2 and C" was quite unlike that on 2328. In spite of the extremely high grass count, the infection was quickly terminated and when the animal was killed on July 30th no lungworms could be recovered from it. It seems unlikely that this difference should be due to an inherent individual insusceptibility on the part of the calf when it is considered that it was exposed to a grass count of the order of 340 larvae per pound, and infestation some 30 or 40 times the lethal level.

A calf, 2466, put in on May 25th, just as the grass count began to rise steeply, died from acute parasitic bronchitis after only 29 days.

Enclosure "A" (Fig. 4) was allowed to grow and was mown for hay June 16th, 1952. It will be seen that on "A" a measurable infection lasted for longer than on the other paddocks and it is particularly noteworthy that the grass count rose when the hay was cut. This was presumably due to the fact that the larvae were situated low down on the herbage, and they were more dilute in that portion which was removed in hay making than in that which was left. As soon as the hay had been carried off and there was a sufficient growth of aftermath, a calf, No. 2233, was put in. This acquired only a light lungworm infestation in the first instance but its faecal larval count began to rise towards the end of August, presumably as the larvae that it passed as a result of the initial low grade infection began to become available on the ground. The calf scoured violently throughout its time on the paddock and its death was precipitated by phenothiazine poisoning on September 20th. The sward was very lush and rich in clover. The grass count rose to a height of nearly 70 larvae per pound and fell away very gradually.

The hay was fed to a housed calf, No. 2265, for a period of five weeks from July 4th to August 8th. On August 18th it was killed but no lungworms could be recovered from it, confirming that these larvae do not survive the process of haymaking.

Discussion

This work may be regarded chiefly as serving to indicate the kind of information that may be obtained from such observations and the difficulties that are liable to arise. Of the difficulties perhaps parasitic gastro-enteritis is the most serious. It has, however, been found in a subsequent experiment that weekly doses of phenothiazine very largely help to check the build-up of stomach and intestinal worms on the paddock without appearing in any way to influence the lungworms. Moreover, if stomach and intestinal worms are held in check in this manner and the calves never become clinically affected with parasitic gastro-enteritis, the danger

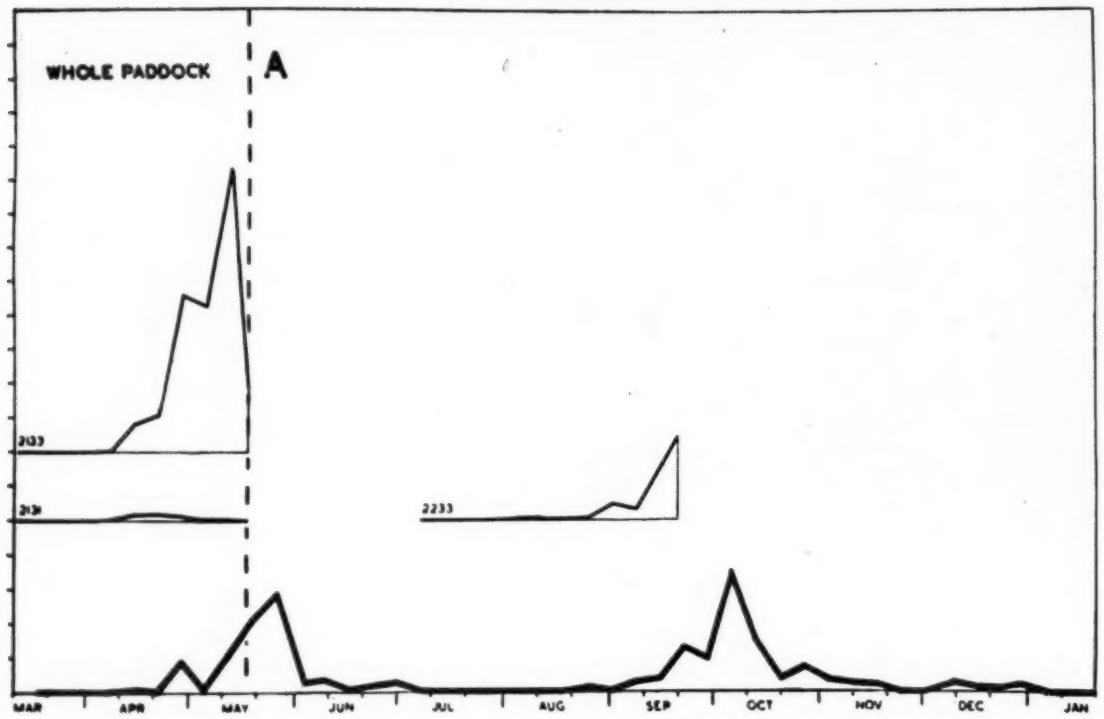

FIG. 4.—The course of the infection on A and in the calf grazing on it. One division on the vertical scale represents 20 larvae per lb. of herbage or 50 larvae per gramme of faeces.

from phenothiazine poisoning does not appear to arise. It has been our experience that calves suffering from acute parasitic gastritis are peculiarly susceptible to the toxic effects of phenothiazine.

Besides points of this nature, however, the results obtained clearly indicate the great possibilities of epidemiological experimentation. Another experiment is already in progress, and more are planned. At the same time a number of tentative conclusions relative to the epidemiology of the disease may be drawn.

Thus it is clear that the level of infestation on the herbage largely determines the effect on the grazing calf and the evidence seems to suggest that old larvae are less infective than young ones.

While there is a tolerably close correlation between the grass count and the severity of the infection produced in a calf, the connection between the numbers of larvae being passed on to the ground and the grass count produced, is, however, a little less close. Winter would seem to offer unfavourable conditions for the translation of faecal larvae into herbage infestation. In spring and early summer, however, unexpectedly rapid build-ups may occur in the herbage. The high levels at this time of year, however, are short lived, for it appears that in summer the infestation on the herbage falls away much more rapidly than it does in autumn. The observations on "D" would suggest that the five and a half months that this piece was empty were sufficient for the infection to die out. This single observation is supported by the result of a more recent experiment. On November 17th, 1953 a calf passing *D. viviparus* larvae was put on a small paddock, one-eighth of an acre in extent, which was enclosed by a double fence. On December 7th, 1953 it was removed and the paddock remained empty until May 19th, 1954 when two young worm-free calves were put in. When these were slaughtered on June 25th, 1954 no worms could be removed from their lungs. These results do not, of course, imply that the larvae will not live through the winter under any circumstances.

Under the circumstances of these experiments it is probably safe to say that a piece of ground that has been free of cattle for four autumn months or two summer months is unlikely to have upon it a dangerous infestation, though it may be that particularly favourably conditions, such presumably as might be found in dense shade-producing herbage, might well extend these periods. The time taken for every single larva to have disappeared is more difficult to estimate but we would suggest that six winter months or four summer months may in practice prove to be a reasonable formula.

An interesting circumstance for which at present no explanation can be suggested is the appreciable time lag between a peak of faecal larval counts and the corresponding one of grass count. In some cases this happened even though the source of infection died or was removed as soon as it reached its peak of

FIG. 5.—Infective larva of *D. viviparus* more than three weeks old.

larval output. This time lag is about a week longer than could be accounted for by the time taken for the larvae to develop to the infective stage.

The picture that emerges of the epidemiology of husk is markedly unlike that of parasitic gastro-enteritis. The infestation on the herbage, which largely determines the clinical effect produced upon a susceptible animal, is subject to wide fluctuations which may be extremely rapid. At the same time the range between a harmless grass count and a lethal one is very narrow. The level of infestation on the ground is determined only in part by the number of larvae being passed on to it in the faeces of infected animals. Other factors probably act by influencing the micro-climate amongst the herbage. Thus, suitable conditions for the creation of high levels of pasture infestation may occur in spring and early summer and also in autumn, but while such high levels are short-lived in spring and summer they persist in the autumn. Lush herbage, especially if the animals are scouring, appears to help small numbers of larvae in the faeces to give rise to high grass counts and it may be that the increasing prevalence of such conditions account for the impression that outbreaks of husk occur earlier in the year than they used to.

If we visualise the infection as being carried through the winter in animals on those farms where the stock is housed, or both on the pasture and in the animals where the stock run out in winter, then the infection must start each year from small beginnings. It appears to be possible for a build-up of infection as between the herbage and the animals to occur in the case of young calves and an outbreak will ensue when the infestation on the herbage has reached a sufficient level. This will occur earlier and be of greater severity if the build-up of infection is rapid. As has been shown, fluctuations in grass counts can be very rapid indeed and it must be accepted that an animal passing only small numbers of larvae is potentially capable of producing lethal herbage infestations.

Summary

(1) There is a close correlation between the number of larvae per pound of herbage and the clinical effect produced on a susceptible calf.

(2) The infestation on the herbage is subject to wide fluctuations which may be extremely rapid. Meanwhile the range between a harmless herbage infestation and a lethal one is very narrow.

(3) The concentration of larvae on the herbage is affected not only by the numbers passed on to the ground in the faeces but to an even greater extent by climatic and other factors. Under favourable circumstances faeces containing only small numbers of larvae may give rise to a high level of infestation on the herbage.

(4) The concentration of larvae on the herbage falls away quickly in spring and early summer but the larvae may persist for four months in autumn and early winter. The infestation of pastures may completely disappear during the winter months.

Acknowledgment.—We are indebted to Dr. E. L. Taylor for his unfailing support and encouragement.

References

TAYLOR, E. L. (1939). *Parasitology*. **31**. 473-478. Technique for the estimation of pasture infestation by strongyloid larvae.

PARFITT, J. W. (1955). *Laboratory Practice*. **4**. 15-16. Two techniques used for the detection and enumeration of the larvae of *Dictyocaulus viviparus* in faeces and herbage.

FIG. 6.—Infective larva of *D. viviparus* up to two weeks old.

Hexamitiasis of Turkeys

BY

J. E. WILSON and D. SLAVIN

Lasswade

AN enteric disease of turkeys caused by *Hexamita meleagridis* is an important source of loss to the turkey rearing industry in some parts of the United States of America both on account of mortality and wastage through failure of affected turkeys to develop normally. In the past the disease has been diagnosed at this laboratory on several occasions, but during the last two years several very extensive and serious outbreaks have been investigated. It is possible that the disease is more prevalent than has been suspected, for due to certain difficulties in demonstrating the parasite in dead birds it may have been unrecognised.

With the rapid and still growing expansion of the turkey industry in this country it is probable, however, that hexamitiasis will become more common and assume a greater importance. No detailed account of the disease appears to have been published in Great Britain; the present paper has been written to record our experience of the disease in the hope that it may prove helpful to research workers and to veterinary surgeons in practice who may be called upon for advice.

Historical

It was shown by Hinshaw, McNeil & Kofoid (1938) and Hinshaw & McNeil (1939) that turkey enteritis was caused by *Hexamita meleagridis*. The morphology of the parasite is fully described in a later publication (McNeil, Hinshaw & Kofoid, 1941). The disease has also been recorded by Jungherr & Gifford (1944) and by Doyle, Cable & Moses (1947). In this country where, except in certain fairly well defined areas, until recently there has been no specialised turkey rearing industry comparable to that in the U.S.A. there are few authenticated records of the disease. Campbell (1945) reported the presence of *Hexamita* in turkeys infected with *Cochlosoma* but in this outbreak it appeared that the latter parasite was the primary cause of disease. In a paper by Slavin & Wilson (1953) a description is given of the morphology and life history of the parasite; this contained some hitherto unrecorded information and differed in certain respects from results already reported.

Species Affected

Turkeys are most susceptible from a few weeks old up to about 10 to 12 weeks but occasionally day-old poults may become infected as soon as they leave the incubator. Adult birds which have survived an outbreak may be carriers.

There are a number of records in American literature of the recovery of *Hexamita* from wild birds. McNeil, Platt, & Hinshaw (1939) found *Hexamita* in quail and in chukar partridges. Hinshaw & McNeil (1941) examined several species of game birds and other wild birds killed on or near infected premises but failed to demonstrate the organism. In a later survey it could not be demonstrated in chickens, ducks, pigeons or guinea fowls. The parasite was reported in pea fowls (California State Department of Agriculture) and in pheasants by Hinshaw & McNeil (1942) and Stover (1943). We have failed to find it in chicks, sparrows, pheasants, rats, and mice from the premises where our outbreaks occurred.

Spread of the Disease

Recovered birds may become carriers and the disease is usually spread to poults by food becoming contaminated by the excreta of such birds. As the flagellate stage of the parasite disintegrates almost immediately it comes into contact with water it appeared to us unlikely that the drinking water was a source of spread of infection. The discovery of a true cystic stage of the parasite present only in the intestinal mucus has altered this view. This stage is extremely resistant and the tenacious character of the mucus enables it to adhere to the boots, clothing, and hands of attendants and to utensils and equipment. It appears to us as the likeliest and most important method of spread of the disease. It has been suggested that a build-up of infection is usually necessary for the precipitation of an acute outbreak and in one of our cases this appeared to be so, for it was not until the last batch of the season that a serious outbreak occurred resulting in 100 per cent. mortality of the affected poults.

That a build-up is not always necessary was demonstrated in another outbreak where the disease occurred in a group of poults within seven days of their being transferred to a large building, part of which had housed the survivors of an outbreak of the previous year. This particular group of poults was the first of the season to be placed there. The disease occurs under all systems of husbandry. Although birds running on solid floors are perhaps generally the worst affected, severe outbreaks with heavy mortality can occur in birds running on wire floors and in tier cages.

Symptoms

The severity of the symptoms depends on the age of the poults affected and the weight of infection to which they are exposed. In young birds mortality may be very great, reaching up to 100 per cent. In older birds outbreaks are usually milder and mortality may be low. This is not always so; the worst outbreak we have seen occurred in birds three to four months old.

In natural cases symptoms usually appear six to seven days after infection is picked up, but in experi-

mental transmissions, using cysts as inoculum, symptoms appear within three days of direct oral infection by stomach tube. There is slight ruffling of the feathers, lessened activity, drooping of the wings and listlessness. Even before any of these symptoms become evident there is usually alteration in the faeces. This is particularly noticeable where birds are kept on wire floors when one or two greatly altered "droppings" are seen among the healthy faeces. Droppings are liquid and slimy, greenish yellow in colour sometimes splashed with brown. Later they become still more fluid and frothy. They have a foetid odour and it is claimed by owners of turkeys who have had experience of the disease that this is sufficiently characteristic to provide a means of identification.

In mild outbreaks only a small proportion of the birds may be affected; few deaths occur and the remainder recover. In more severe outbreaks, however, every bird in the pen or even in whole sections of the flock may be affected. Symptoms develop rapidly. Birds become increasingly inactive. When made to walk they do so with a stilted gait and appear to lift their legs higher from the ground. The back is always arched and the wings droop (Fig. 1). Appetite is lost early in the course of the disease but thirst is marked for a time.

FIG. 1.—Hexamitiasis. 10-week-old turkey. Ruffled feathers, arched back, drooping wings.

Listlessness increases and birds stand motionless, huddled in corners with eyes closed, heads lowered or tucked under the wing, backs arched, wings slightly protruding from the body and drooping, presenting a picture of abject misery (Figs. 2 and 3). Some may remain on the perches, others on the floor. They neither eat nor drink and the absolute silence in a large house containing hundreds of turkeys, all completely immobile, is a striking and unforgettable feature. The silence is most noticeable to anyone acquainted with the "noise" always present where large numbers of turkeys are housed.

Bodily condition is lost very early in the disease and this becomes more rapid as the disease progresses. Dehydration is one of the most striking

FIG. 2.—Hexamitiasis—advanced. 14-day-old poult.

FIG. 3.—Terminal stages of Hexamitiasis. 12-week-old turkeys.

symptoms. The skin becomes dry and very adherent to the underlying muscle. This is particularly evident over the breast muscles and on the legs. The shanks lose their rounded appearance, becoming flattened laterally. As a result of shrinkage the tendons below the hock stand out more prominently and the back of the shank presents a sharp, ridge-like appearance (Fig. 4).

The disease spreads with great rapidity. At first only a few birds in a large group may be seen to be affected, yet within 24 to 48 hours almost every bird in the pen may show symptoms.

FIG. 4.—Shank showing dehydration and prominent tendons.

Pathology of the Disease and Post-Mortem Appearance

A constant *post-mortem* finding in birds which have died of the disease or which have been killed late in its course is the marked dehydration of all the body tissues. This is particularly noticeable in the breast muscles which are shrunken and darkened and from which the skin can be removed only with difficulty. The condition of the shanks has already been described. All the tissues feel tacky to the touch and when the intestines are being removed they tend to stick to the fingers if handled. In birds more than a few weeks old the intestines are slightly hyperaemic but in very young birds this is less evident. In freshly killed severe cases the intestine shows numerous areas of contraction, the bowel wall is generally thickened throughout especially in older birds, and when opened it frequently everts along its length and may turn completely outside in. The entire small intestine is filled with watery, frothy, clear mucus in which only occasional strands of greenish food matter can be seen. The bowel fluid shows enormous numbers of flagellates and generally many macrophages. The caeca are filled with watery, yellow, frothy faeces.

The essential lesion is the invasion of the reticulo-endothelial cells and tissue interspaces by developmental forms of the parasite. This occurs throughout the small intestine and rectum but has never been seen in the duodenum or caeca. The earliest invaded sites are the terminal portion of the small intestine and rectum and the first schizonts and flagellates have usually been seen there. This may be related to the time required for the cysts to develop invasive forms in their passage through the intestine. In cases killed 72 hours after artificial infection, *i.e.*, before the flagellate stage in the cycle has been reached, the early schizonts can be seen between the epithelial cells, in the tissue interspaces of the villi and in the reticulo-endothelial cells of the lower small intestine and rectum, but as the disease progresses the entire small intestine is involved. Schizonts appear quite capable of growth in the tissue interspaces but development to the motile form is generally within the reticulo-endothelial cells; in advanced cases these cells are packed with developing forms (Fig. 5).

FIG. 5.—Reticulo-endothelial cell free in the bowel, containing developing parasites x 1500.

The epithelial cells at the top of the villi are filled with mucin and generally raised from the basement membrane, with various sized dark-staining globules between the basement membrane and the epithelium. Even the most advanced cases when examined fresh have shown little desquamation of epithelium or haemorrhage. Any marked desquamation can be attributed to *post-mortem* change. The crypts of Lieberkuhn are packed with flagellates and when one considers the numbers to be seen in a 5.0μ section of a single crypt, adds the numbers which further sections of the same crypt would show, multiplies by the number of crypts in the bowel and then considers the numbers of developing forms still within the cells of the villi, one gets some conception of the enormous parasitic load carried by an infected poult and its chances of survival (Fig. 6).

As is shown in our description of the life cycle (Slavin & Wilson, 1953) the initial development of the parasite in a new host is directed towards the establishment of a population and the schizogony cycle is well advanced when the birds first show symptoms of illness. We have repeatedly examined early cases in which the concentrated washings from the entire small intestine and numerous wet scrapings failed to show any flagellates yet stained smears showed numerous merozoites and schizonts throughout the intestine.

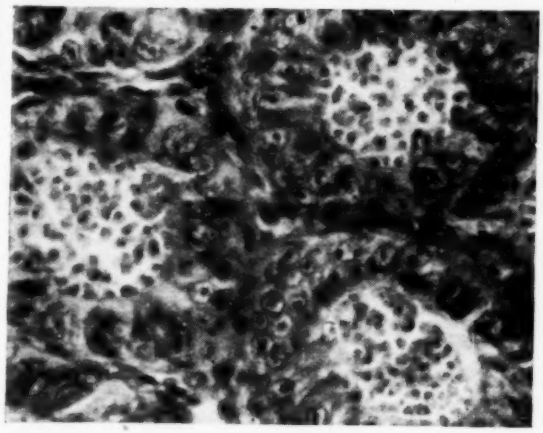

FIG. 6.—Cross section of base of crypts showing enormous numbers of mature flagellates x 900.

Diagnosis of the Disease

Where there has been a history of heavy losses among young turkeys up to three months old with a high simultaneous morbidity rate and symptoms of mopishness and diarrhoea, hexamitiasis should always be suspected. When material from such a source is received in the course of ordinary *post-mortem* examinations and the carcases show advanced dehydration with shrinkage and darkening of the breast muscles and there is difficulty in flaying the skin, and the tissues in general feel tacky, a diagnosis of hexamitiasis is almost certain to be correct. Such material is not generally suitable for demonstration of the flagellate or its developmental stages as these rapidly disintegrate *post-mortem*. In some cases we have failed to demonstrate any parasites in birds which could not have been dead for more than three hours. Therefore the only means of diagnosing the disease with certainty is by examination of freshly slaughtered birds. If a drop of bowel fluid or a wet scraping is examined under the ordinary microscope with the light suitably reduced, there should be no difficulty in seeing the flagellates, especially if the slide is heated to body temperature to make them actively motile.

However, in the first few days of an outbreak we have frequently found that only merozoites and schizonts are present and for their demonstration a dry smear stained by one of the Romanowsky modifications is essential. In our experience the best method is to take several impressions of cross sections of small intestine, dry them rapidly with the blast from a hair drier, and stain with MacNeal's Tetrachrome. The rapid drying gives a much superior picture to ordinary air drying. In the very early stages of the disease merozoites and schizonts are most easily found in the terminal portion of the small intestine and rectum but generally, in birds killed before the appearance of the flagellates, they are to be seen throughout the small intestine.

When fresh wet preparations from advanced cases are examined under the ordinary microscope, but especially when examined under phase contrast, there should be no difficulty in identifying *H. meleagridis*. It is 7.0μ in length by 4.0μ to 4.5μ in breadth and has a lanceolate outline, the greatest breadth being slightly behind midline of the parasite's length and the fore end being sharper than the tail end. There are eight flagella, all of them arising from the anterior nuclear chromatin. The six anterior flagella emerge in groups of three from either side of the head while the two posterior ones pass down the body of the organism and cross over at about two-thirds of their length to emerge on either side near the posterior end. In stained preparations the parasite presents a somewhat truncated appearance with broader extremities (Fig. 7).

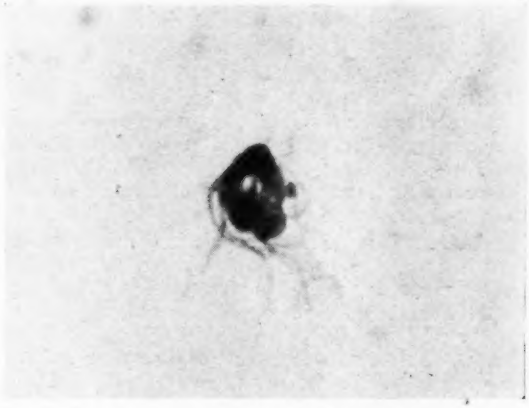

FIG 7.—Mature *Hexamita* showing eight flagella stained smear x 3000.

The flagella have not been measured but are approximately twice the length of the body. At the cross-over of the axonemes of the posterior flagella there are always either four or eight chromatin dots. These are well shown in Fig. 8, while the chromatin structure of the nucleus is shown in Fig. 9. Both prints have been purposely under-exposed

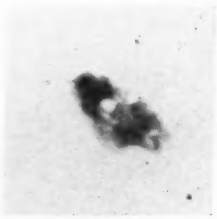

FIG. 8.—Under developed print to show the chromatin dots at the cross-over of the axonemes in the mature flagellate. Stained smear x 1500.

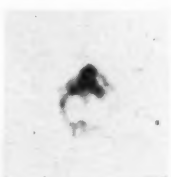

FIG. 9.—Under developed print to show the chromatin structure of the nucleus in a mature flagellate. Stained smear x 1100.

to bring out these details at loss of definition of other structures.

The mature flagellate is very actively motile on a warm stage and progress by a zig-zag, darting movement; all have a variable number of vacuoles. The only parasite with which it could possibly be confused is a Trichomonad which was found here in some cases of acute diarrhoea in young poults; the two were easily distinguished under phase contrast and as the Trichomonad appeared to cause little upset except when present in enormous numbers it was not studied further. It was found only in the latter end of the small intestine and caeca.

Some Aspects of the Life Cycle of *Hexamita meleagridis*

Workers on hexamitiasis in turkeys have generally had difficulty in explaining the source of a fresh infection in young birds; vectors such as flies and vermin, and the transport of infection on clothing and utensils, have all been suspected. There is no difficulty in tracing the spread of infection once an outbreak has started.

A close study of the life cycle in the host has been made by one of us (D. S.). In the fluid droppings, and in any incubated broth suspension of the flagellate, what in an earlier publication we have termed a "cyst" can be demonstrated (Slavin & Wilson, 1953). This cyst was found to have poor resistance to external environment and was readily destroyed in distilled water. It disappeared from broth suspensions when they became heavily contaminated with bacteria. It did not resist drying and it was, therefore, unlikely to survive on the surface of an egg during incubation or in the dried dust from infected droppings.

Survival of this cyst form was not studied extensively but fluid faecal material in which only this form could be seen will infect poults after it has lain one week on the bench at room temperature, or six weeks at 4° C. Droppings kept over two months at 4° C. did not infect. These cysts are present in enormous numbers in the fluid droppings, but after a few days at room temperature there is a gradual reduction in their number and after a week they may be difficult to find in wet preparations.

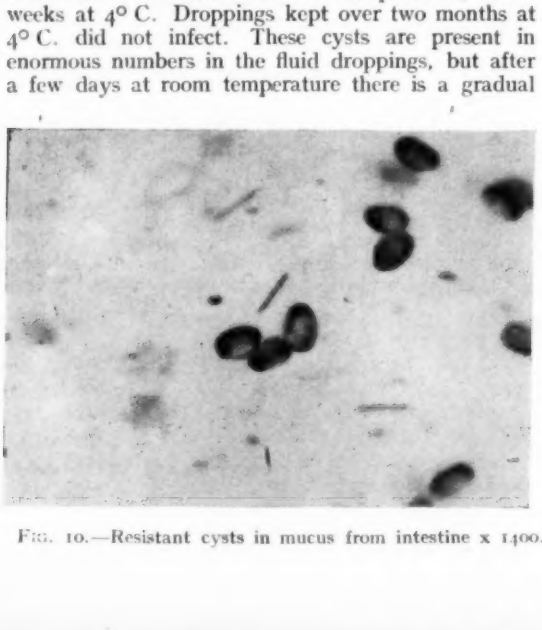

FIG. 10.—Resistant cysts in mucus from intestine x 1400.

In our outbreaks the greatest losses occurred under intensive systems of turkey rearing. In every one the eggs were fumigated before incubation and the poults were kept on wire and well isolated from adults until about 12 weeks old. Whether turkeys or fowls are being reared, the use of wire floors and isolation from adults have proved themselves efficient controls at least for a time against all other intestinal infections which do not pass in or on the egg. But in hexamitiasis, even when wire floors have been continually used, this is not so and the general picture is that a flare-up of the disease occurs in poults a fortnight to three weeks old. The work here has so far not furnished an explanation for this.

In all acute cases of hexamitiasis large amounts of watery mucus and variable amounts of clotted mucus are present in the intestine. At the beginning of this year a study was commenced on the development stages of the flagellate in the clotted mucus. Certain forms were found which will be described later; these included an important form which provided an explanation of most of the puzzling features present in a fresh outbreak of hexamitiasis. This is a resistant cyst (quite distinct from the so-called cyst previously described) which has been found only in the clotted mucus. It has not been seen in the fluid mucus or free in the fluid droppings and hence was missed in previous examinations. It is found in enormous numbers in the flecks of mucus in fresh droppings from acute cases indicating that encystment takes place in the bird.

The resistant cyst is a symmetrically oval body 6μ to 7μ long by 4μ to 5μ wide with blunt rounded ends (Fig. 10). In the resting form there is a neutrophilic cyst wall about 0.5μ thick. By the position of the nuclear material at one end and the crossover of the axonemes at the other it can be determined that each cyst contains a single flagellate. When wet preparations are examined on a warm stage by phase contrast the flagellate can be seen encysting. It makes its way into the thick strands of mucus, spinning around in various directions, then slowly loses motility, and assumes a symmetrically oval shape and gradually a cyst wall is developed. In wet preparations of fully developed resting cysts no internal structure can be determined but the cyst wall is clearly seen. Sluggish movement of the flagella, which appear to be wound transversely within the cyst is occasionally noted. The actual time required for the development of the resistant cyst from the flagellate has not been definitely determined but it is short.

The release of the flagellate from the cyst has been clearly seen by taking flecks of clotted mucus from the fluid droppings of a poult shortly after being killed. These were put under a sealed coverslip in 5 per cent. albumin broth and examined by phase contrast on a warm stage. Resistant cysts and flagellates were present in enormous numbers. After two hours the cysts showed a small projection at one end. These forms were at first thought to be incompletely encysted flagellates but after three and

(Continued on page 241)

a half hours the projections were half the length of the cyst and could clearly be identified as emerging flagellates with six anterior flagella. After five hours a flagellate was seen to leave a cyst and became very actively motile immediately on release.

The process of liberation is as follows: when resting mature cysts are put into a fresh medium and incubated there is at first a sluggish movement of the flagella which are wound around the parasite within the cyst wall. The head then protrudes through one end of the wall and the flagellate gradually emerges through the narrow opening, having to constrict itself considerably in the process. When the parasite is about a third to one-half emerged, the six anterior flagella can clearly be seen moving within the cyst wall. All are of the same length as in the mature flagellate. Because of the narrow opening, release of the parasite from the cyst is slow and laboured and some have been seen struggling to free themselves with only a fraction of their bodies retained as a small blob within the cyst. Once the flagellate has freed itself it becomes very actively motile and is quickly out of view.

The further stage of development of the flagellate has not been determined but seems likely to be the so-called "cyst" form previously described. These forms can be readily produced from mature flagellates by simple incubation at 37° C. in broth and from them probably develop the invasive forms. Fuller descriptions will be given in a future publication.

The true resistant cyst is found only embedded in the clotted mucus so the latter must contain some substance necessary for the elaboration of the cyst wall. No method has been found of freeing the cysts from the mucus. When flecks of infected mucus were left 24 hours in distilled water the cysts remained morphologically intact. They also remained intact when dried in a petri dish overnight. The fact that the resistant cyst develops only in the clotted mucus gives it a valuable aid to survival, as the mucus provides great protection against adverse environment, especially drying. The viability of the cysts left in distilled water or dried in mucus has not been tested by transmission experiments but there seems no reason to think they should not remain infective as they are still structurally intact and morphologically identical with viable cysts after these treatments. This work had to be stopped temporarily as the strain of *H. meleagridis* used in transmission died out, but it is now continuing.

Persistence of infection in birds recovered from acute hexamitiasis has not been studied extensively here, but a few recovered birds have died of the acute disease up to three months after infection. American workers found that up to 30 per cent. of recovered birds became carriers. In acute exacerbations of the disease in carrier birds the picture is exactly the same as that in acute infections in young birds. Birds up to a few weeks old can tolerate large numbers of parasites without much apparent upset to their health and it is reasonable to assume that this is the case also in adults. It is the cysts from these cases which are responsible for fresh outbreaks of the disease.

Control and Prevention

Therapeutics. Every anti-protozoal drug available commercially in this country (including anti-malarials, trypanocides, amoebicides, and anti-luetics) and others which were supplied for experimental trials were tested *in vitro* and *in vivo*. A few destroyed the flagellate form of the parasite free in the intestinal tract but none had any effect on the invasive stages in the bowel wall. It should be pointed out that a highly virulent strain of *Hexamita* was used in these experiments and birds were given a lethal dose. The copper sulphate-whey treatment described by McNeil (1948) was not found of any value, nor were antibiotics, which, however, were not tested *in vivo*. 2-amino-5-nitrothiazole (better known as Enheptin in America and Entramin in Great Britain) has been partially successful but never more than 50 per cent. of artificially infected poults recovered after treatment with it.

Di-n-butyltin dilaurate (Tinostat) was tested in natural and experimental outbreaks. In the field it appeared to control mild outbreaks and there was improvement in the appearance of treated birds and a lowered death rate in more severe outbreaks. It had no apparent effect, however, when given to birds experimentally infected with a lethal dose of the parasite. Its action is probably direct on the free flagellates in the bowel and it may well be more effective when used as a prophylactic, as directed by the manufacturers who recommend that poults should be given the medicated mash "before an outbreak is expected and continued past the danger period" (during the first 10 to 12 weeks).

It appears to us that as regards curative treatment any drug likely to be successful would require to be administered in the drinking water, as affected birds cease feeding so early in the course of the disease; food medication might be possible for prophylactic purposes.

It was patent that the use of any drug requiring individual handling of sick birds is contra-indicated; death rates in such cases were invariably higher in treated groups than in control pens.

Hygienic Measures. Growing poults and older stock should be kept as far apart as possible and every effort should be made to prevent all contact, direct or indirect; poults are vulnerable from hatching until three to four months old. Care should be taken to ensure that foodstuffs do not become contaminated with excreta from affected birds or carriers. A separate food store for growing stock is desirable and if separate attendants cannot be provided the most stringent precautions should be taken if the disease is suspected. Hands should be washed before commencing feeding and boots should be thoroughly cleansed before entering the rearing house. Separate utensils and tools should be used and droppings, floor sweepings, etc. should be placed in a separate site to prevent any possibility of contamination from the droppings of adult birds, which could readily occur if a common manure barrow or pit were used.

Wherever possible affected poults should be destroyed when the first signs of illness become apparent. This may abort a serious outbreak but the onset is so sudden that by the time a diagnosis is obtained (even if this is only a matter of hours) the disease may be widespread in the pen.

As regards disinfection, the flagellate form of the parasite is readily destroyed by any of the usual agents, and the so-called cystic form, as was earlier stated, rapidly disintegrates in ordinary water.

Because of its protective coating of mucus the cystic form now described can probably withstand treatment with the usual disinfectants which may fail to penetrate sufficiently to kill the large numbers present.

It is suggested that thorough cleansing followed by soaking with hot water containing 4 per cent. washing soda or where available, treatment with live steam is likely to be effective.

Summary

The history, symptoms, pathology and *post-mortem* appearances of hexamitiasis of turkeys are described. Attention is drawn to some hitherto undescribed aspects of the life history of the parasite.

Therapeutic treatment has proved disappointing. 2-amino-5-nitrothiazole has been partially successful. Di-n-butyltin dilaurate (Tinostat) appears to be the most promising drug so far tried.

Acknowledgments.—Thanks are due to the various drug firms who supplied material for test, and to Dr. J. E. Salsbury, of Charles City, Iowa, U.S.A. for providing the Tinostat used in the trials.

References

CALIFORNIA STATE DEPARTMENT OF AGRICULTURE. (1945). "Hexamitiasis in Peafowl." *Ann. Rep. Petaluma Poultry Path. Lab. Calif. St. Dept. Agr. Monthly Bul.* **30,** 455.

CAMPBELL, J. G. (1945). "An Infectious Enteritis of Young Turkeys Associated With Cochlosoma sp." *Vet. Jour.* **101,** 255.

DOYLE, L. P., CABLE, R. M., & MOSES, H. E. (1947). "A Destructive Turkey Disease." *Jour. Am. Vet. Med. Assn.* **111,** 57.

HINSHAW, W. R., & MCNEIL, E. (1941). "Carriers of Hexamita meleagridis." *Am. Jour. Vet. Res.* **2,** 453.

——— ———. (1942). "Hexamita sp. from the Ring-Necked Pheasant." *Jour. Am. Vet. Med. Assn.* **101,** 503.

JUNGHERR, E., & GIFFORD, R. (1944). "Three Hitherto unreported Turkey Diseases in Connecticut: Erysipelas; Hexamitiasis; Mycotic Encephalomalacia. *Cornell Vet.* **34,** 214.

MCNEIL, E. (1948). "Hexamitiasis of Turkey Poults." *Calif. Agr.* **2(11),** 15.

MCNEIL, E., PLATT, E. D., & HINSHAW, W. R. (1939). "Hexamita sp. From Quail and From Chukar Partridges. *Cornell Vet.* **29,** 330.

SLAVIN, D., & WILSON, J. E. (1953). "Hexamita Meleagridis." *Nature.* **172,** 1179.

STOVER, D. E. (1943). "Hexamita sp. From the Ring-Necked Pheasant Transmissible to Turkeys." *Jour. Am. Vet. Med. Assn.* **103,** 37.

Current Literature

ABSTRACTS

A Peculiar Nutritional Dermatitis in Pigs. THOMAS, G., & EDEN, E. (1954). National Agricultural Advisory Service. *Nature* (1954), **174.** 553.

The authors have often encountered a dermatitis in pigs of 10 to 12 weeks of age. Initially there are red blotches under the skin of the hocks and these spread later up the hind legs into the groin and over the belly and eventually over the back and ears. There is marked pruritis and as the pigs rub themselves the skin breaks and there is extensive scab formation. Appetite is poor and consequently there is little or no live-weight gain. Natural recovery is slow.

The condition develops in pigs on both home-mixed and proprietary rations, with or without antibiotics. It cannot be controlled by topical applications of the usual oily and powder dressings or by dipping, bathing and spraying with insecticide preparations. It has only been found when pigs are dry-fed, even though drinking water is freely available.

There is immediate recovery when wet-feeding is instituted. The inclusion of fodder beet or potatoes in the diet results in marked improvement but this is not nearly so dramatic as with wet-feeding. The authors cannot offer any explanation as to the fundamental cause of the condition or give a valid explanation of the mechanism of the empirical cures but they put their observations on record in the hope that it will help others who have encountered the condition.

S. J.

Fluorosis in Farm Animals. ALLCROFT, RUTH. (1954). *State Vet. J.* **9.** 1.

Fluorosis in this country arises from the fluorine contained in industrial gases. The fluorine content of soil, water, plants and animal tissues is given and the species' susceptibility of the various species following the intake of high doses is listed as calves > milk cows > other bovines > sheep > pigs > horses > poultry.

The principal clinical sign of fluorine poisoning in cattle is lameness associated with skeletal abnormalities. On *post-mortem* the bones appear chalk-white with surface irregularities, a thickened peritoneum and degenerated bone marrow.

Diagnosis should be confirmed by an estimation of the fluorine content of bone, in the case of dead animals, or urine from live ones. In view of the great contamination of grass near sources of fluorine, the determination of its fluorine content is useful corroborative evidence.

The total loss in this country is not great but the loss to certain farmers is considerable. The industries investigated included aluminium, brick, glass, iron and steel works, power stations and potteries.

Control suggested is by preventing atmospheric pollution with appropriate smoke traps and by limited grazing.

Acquired Toxoplasmosis. BEVERLEY, J. K. A., SKIPPER, E., & MARSHALL, S. C. (1955). *Brit. med. J.* March 5th, 1955. 577-578.

Although it has long been realised that toxoplasm infests many species of animals, including domestic, direct transmission of the disease from animal to human has never been conclusively demonstrated. Transmission from human to human is known to occur only in the case of a woman who bears a toxoplasmic infant.

One interesting suggestion made in 1954 was that one form of transmission from human to human may well be direct contact spread through saliva.

The subject of this report on acquired toxoplasmosis was a laboratory worker who accidentally pricked his finger with a needle that had been used to fill a syringe with mouse peritoneal exudate rich in the Rh strain of toxoplasma. Nine days later he became ill and in spite of penicillin therapy the temperature ranged between 100° and 103° F. A week later a five day course of sulphadimidine was commenced and in 10 days progress towards recovery was noted. There are six cases of toxoplasmosis in laboratory workers recorded in literature between 1951 and 1953.

G. F. B.

Treatment of Acute Rheumatic Fever in Children. A Co-operative Clinical Trial of A.C.T.H., Cortisone, and Aspirin. *Brit. med. J.*, March 5th, 1955. 555-574.

This is a joint report of work done at six centres in the United Kingdom, five in the United States and one in Canada which was designed to compare the relative merits of A.C.T.H., Cortisone and Aspirin in the treatment of acute rheumatic fever and the prevention of rheumatic heart disease. The present report relates to children under the age of 16 and compares the effects of the three drugs in the acute course of the disease and in the persistence and development of rheumatic heart disease through one subsequent year.

It is nearly six years since reports were published that adrenal cortical hormone gave immediate and gratifying results in the treatment of acute rheumatic fever, and subsequent reports have borne witness to the efficacy of adrenal hormone during the acute stage of rheumatic infection. The question however, remained unsolved as to whether it is a better treatment than salicylates, and whether it does possess the additional advantage of preventing progressive and permanent cardiac damage.

This co-operative clinical trial involving 497 patients (240 in United Kingdom and 257 in United States and Canada) goes some way to answering these questions.

The trial produced no evidence that any of the three agents resulted in uniform termination of the disease and in all treatments some patients developed fresh manifestations during treatment. Treatment with either of the two hormones resulted in more prompt control of certain, but not all, acute manifestations, but this more rapid disappearance was balanced by a greater tendency for acute manifestations to re-appear for a limited period upon cessation of treatment. At the end of one year there was no significant difference between the three treatment groups in the status of the heart. While either of the two hormones gave a more rapid change in the atrioventricular conduction time at the end of 63 days the position in regard to all three treatments showed no difference. Either of the two hormones gave a more immediate change in the erythrocyte sedimentation rate but at the end of 42 days when treatment was stopped all three treatments had produced similar changes. Within a week after stopping treatment there had been a marked deterioration in those cases receiving either hormone. No such deterioration took place in the patients receiving aspirin. At the 91st day the position in regard to all three treatments showed no difference.

G. F. B.

(The chief interest in this paper for readers of THE VETERINARY RECORD is that it emphasises how easy it is to form a favourable but unfounded opinion as to the value of some new form of treatment. It illustrates how such false impressions can only be avoided when carefully planned clinical trials involving a sufficiently large number of observations are carried out.)

Cardiac Arrest of One Hour Duration in Dogs Followed by Survival. GOLLAN, FRANK, Thayer Veterans Administration Hospital, Nashville, Tenn. (1954). *Vet. Med.* **49.** 440.

In a series of experiments to determine the cause of death when body temperature falls below 20° C., blood was withdrawn from dogs, oxygenated, refrigerated and recirculated with an artificial heart and lungs. Regular heart beats ceased when the body temperature reached 13° C., but even after one hour at 0° C. with complete cardiac arrest, on rewarming the blood the normal heart beats returned at temperatures of 10° to 15° C. After a half-hour of rewarming the heart reached a temperature of 30° C. and mechanical circulation was stopped. All dogs survived without any detectable damage to heart and brain.

These experiments show that heart failure must be the cause of death in exposure to extreme cold, and they may also have a practical application as a method of arresting the heart for the performance of cardiac surgery.

A. M. L.

BOOK REVIEW

Microbiology of Meats. By LLOYD B. JENSEN, PH.D. Third edn. 422 pp. The Garrard Press, Champaign, Illinois, U.S.A. 1954. Price $6.00.

The author of this work, on the industrial microbiology of meat foods, is the chief bacteriologist in the research laboratories of Messrs. Swift & Co., the noted meat purveyors and exporters of Chicago. He was formerly a lecturer in the U.S. Army Medical Service and he records the debt owed to veterinarians whose long and wide experience in the U.S. Federal Meat Inspection Service and the Bureau of Animal Industry "has resulted in great benefit to the science of meat sanitation and technology which are reflected in many pages of this book."

The introduction consists of a most readable historical résumé of meat as a food for man, and the developments which followed the growth of refrigeration in relation to the transportation of meat.

In the pages which follow, the principles used in the curing of beef and pork are discussed in the light of bacterial processes, and sausage manufacture is adequately covered in conjunction with the bacterial and chemical changes which may occur.

An important and interesting chapter deals with the *ante-* and *post-mortem* bacteriology of normal tissues of swine—and the many theories relating to souring which have been advanced, *e.g.*, faulty sticking and bleeding, improper chilling, bruising, etc., were investigated. Of special interest, one may cite the work in which *ante-mortem* specimens were taken under strict surgical asepsis from the bone marrow of pigs and the results compared with similar specimens, from the same animals, after slaughtering in the normal manner. Whereas no micro-organisms were isolated from the tissues of the living animal, they were found to be present in the specimens taken after slaughter. Jensen discusses the routes by which this bacterial invasion occurs and postulates the theory that previous work, which gave divergent results, was based upon faulty techniques.

Chapters on the changes which take place in various fats in consequence of chemical and microbial action, the growth, survival and destruction of micro-organisms in frozen meat, chemical and bacteriological processes which may take place in canned meats, and a summary devoted to food poisoning and the care of foods, all make interesting reading.

The literature dealing with the microbiology of meat products is scattered over so wide a field that the need for such information to be collected and discussed must be apparent to all those whose interest lies in a scientific approach to the wider aspects of meat inspection. We recommend this work as a useful addition to the meat and food inspector's library.

ACCESSIONS TO THE R.C.V.S. LIBRARY

The following publications have been received in the library.

ANTIGUA. *Agricultural Department.* Annual report, 1953.

BREIREM, K. *and others.* Forsok over hypomagnesemi og ketosis hos melkekyr Hypomagnesemia and Ketosis in dairy cows. Gjovik, Mariendals, 1954.

CANADA. *Veterinary Director General.* Report, 1953-1954.

DREGUSS, M. H. *and* LOMBARD, L. S. Experimental studies in equine infectious anaemia. Philadelphia, Uni. Pennsylvania Press, 1954.

GREAT BRITAIN. *Ministry of Agriculture and Fisheries.* Breeding for bacon production. London, H.M.S.O. 1953. (Bulletin No. 158).

LONDON. Iodine Educational Bureau. Iodine in foods and feeding stuffs. London, I.E.B. 1955 (Iodine information No. 44).

———. ———. Radiographic contrast media. London, I.E.B. 1954 (Iodine information No. 43).

MANIA, T. N. Les plus importantes unités de mesure de la valeur nutritive des aliments employés en zootechnie. Athens, 1955. (Greek text, English summary.) Michigan State College, 1855-1955.

NEW SOUTH WALES. *Department of Agriculture.* Livestock health and animal husbandry. Report No. 28, 1951-1952.

NEW ZEALAND SOCIETY OF ANIMAL PRODUCTION. Proceedings, 1954.

OHIO. *Agricultural Experiment Station.* Acre yields of beef from corn and meadow crops. 1955. (Research Bulletin No. 753.)

SWAZILAND. *Department of Veterinary Services.* Annual report for the year, 1953.

TRINIDAD AND TOBAGO. *Director of Agriculture.* Administration report, 1953.

UNITED STATES OF AMERICA. *Department of Agriculture.* Breeds of chickens for meat and egg production, 1954. (Farmers Bulletin No. 2065.)

———. ———. Dairy cattle breeds, 1954. (Farmers Bulletin No. 1443.)

———. ———. Goose raising, 1954. (Farmers Bulletin No. 767.)

———. ———. Managing winter sheep range for greater profit, 1954. (Farmers Bulletin No. 2067.)

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th February, 1955		58	—	—	26	—	57
Corresponding period in	1954	14	—	1	50	—	41
	1953	48	—	15	13	—	84
	1952	20	—	5	7	—	47
1st January to 15th February, 1955 ...		151	—	5	111	—	140
Corresponding period in	1954	36	—	3	236	—	170
	1953	174	—	18	146	—	199
	1952	62	—	22	39	—	114

News and Comment

R.C.V.S. BANQUET

The R.C.V.S. are holding a banquet on May 12th at the Dorchester Hotel, London. All members of the profession and their ladies will be welcome, and the price of tickets is 2 gns. per person.

It is requested that applications should be made to the Registrar of the Royal College *after* April 1st. A number of distinguished guests have already accepted invitations, and it would be advisable, therefore, for those who would like to be present to make early application for tickets.

LATE DELIVERY OF "THE RECORD"

For some time past discussions have been proceeding at a high level with the Post Office authorities, in an endeavour to ensure that readers should have their copies on Saturdays. A revised printing schedule has been arranged and the Post Office have quickened up their collection of the journals from our printers.

In order to see whether these measures are proving satisfactory, we should be grateful if readers who are not receiving their journals by Saturday noon at latest would be good enough to communicate with the Editor.

AN ANGLO-DANISH OCCASION

A very distinguished company will be present on April 4th at Apothecaries Hall, when the Wellcome Trustees and the Directors of the Wellcome Foundation Ltd. will entertain to dinner the members of the Carlsberg Foundation and Management of the Carlsberg Breweries of Copenhagen.

These two institutions are for all practical purposes identical in their constitution and aims. Both were created with the object of using for philanthropic purposes the whole of the profits from a continuing industrial concern.

The Carlsberg Foundation is the senior and came into being in the 1880s following the founding of the Carlsberg Breweries by the late J. C. Jacobsen and their extension by his son. These two men conceived the plan of leaving the ownership of their breweries to an independent foundation consisting of five eminent Danish citizens appointed by the Royal Danish Academy of Arts and Sciences. It is the responsibility of these five gentlemen to organise and apply the money they receive from ownership of the breweries for scientific research and the promotion of the arts, for the benefit of the Danish nation.

It may well be that the Carlsberg Foundation provided the inspiration and the model for the creation of the Wellcome Trustees. When Sir Henry Wellcome died in 1936 he vested the holding of the whole of the share capital of his extensive industrial interests in five Trustees who were charged to promote and support medical and other research anywhere in the world, and to create and endow museums and libraries dealing with medicine and its history.

The similarity between the two Foundations has led them of recent years to make contacts for the discussion of matters common to their experience and interests. It is to cement the friendly relations between the two that the Anglo-Danish dinner on April 4th is being held.

Sir Henry Dale, O.M., F.R.S., Chairman of the Wellcome Trustees, will preside, and among those present will be H.E. The Danish Ambassador, H.E. The British Ambassador to Denmark, the Marquess of Salisbury, Lord President of the Council, and the Lord Adrian, President of the Royal Society.

FOOT-AND-MOUTH DISEASE

The area described in the Schedule below will cease to be an Infected Area for the purpose of preventing the spreading of foot-and-mouth disease on March 31st.

County of Oxford

Petty Sessional Division of Banbury and Bloxham—The parishes of Shenington, Epwell, Shutford, Sibford Gower, Sibford Ferris, Swalcliffe, Tadmarton, Milcombe, Wigginton, and Hook Norton, and so much of the parishes of Alkerton and Wroxton as lie to the south and west of the main road (A.422) running from Stratford on Avon to Banbury, excluding the said road.

Petty Sessional Division of Chipping Norton—The parish of Rollright.

County of Warwick

Petty Sessional Division of Shipston on Stour—The parishes of Compton Wynyates, Whatcote, Idlicote, Honington, Brailes, Barcheston, Burmington, Cherington, Sutton under Brailes, Little Wolford, Long Compton, Stourton, and Whichford.

Petty Sessional Division of Kineton—So much of the parishes of Tysoe and Ratley and Upton as lie to the south and west of the main road (A.422) running from Stratford on Avon to Banbury, excluding the said road.

THE ASSOCIATION OF VETERINARY STUDENTS

The Association of Veterinary Students held their 12th Annual Conference at the Veterinary College of Ireland, Dublin, in February. Visitors from all the British colleges attended and several European colleges were also represented.

Professor Nicholson, Principal of the college, in opening the Conference, extended a hearty welcome to the visitors.

Papers were read by the following: M. R. Hartnett, Department of Agriculture, Dublin, on "Veterinary Aspects of Agriculture"; Professor Lamont, on "Government of Veterinary Colleges in Great Britain and Ireland and its Changes in Recent Years"; and M. H. Fussell, of Cambridge University, on "Stress."

The main business of the Annual General Meeting included introduction of the new President, Mr. Donald R. Banks, of Cambridge, and the financial report for the year 1953-54. The meeting was informed that A.V.S. had been granted affiliation to the B.V.A. as a Cadet Branch. It was decided that in future years the Conference would be held during the first week-end in February.

The results of the inter-college games tournaments are as follows:—

Dublin won the N.V.M.A. and Wooldridge Rugby Cups, and the R.C.V.S. Soccer Cup; Glasgow won the Table Tennis Cup; The Amoroso Boxing Cup was not contested for.

The Conference dance proved a most successful function and was enjoyed by all.

The Conference was concluded with a trip to the Irish National Stud, Co. Kildare, where the visitors saw Tulyar and his first Irish-born foal.

THE REGISTER OF VETERINARY SURGEONS

The names of the following gentlemen have been restored to the Register of Veterinary Surgeons:—

Mr. Joseph Patrick Fitzgerald, 73, Orchard Road, St. Annes-on-Sea, Lancs; Mr. Samuel Gourley, 1 & 2, Edward Street, Brighton, Sussex; and Mr. Alfred Bernard Over, 15, Downing Street, Cambridge.

STUDENTS' DONATION TO RED CROSS

As a result of the revue "Take Us From Here," given by students of the R.V.C. at Goring Parish Hall, Goring-on-Thames, on March 9th, 10th, and 12th, a cheque for £50 has been sent to the British Red Cross Society.

All those who took part both in the production and its management and staging are to be congratulated on this excellent result.

PERSONAL

Births

BAXTER.—On February 26th, 1955, to Muriel, (*née* Knox), wife of J. T. Baxter, M.R.C.V.S., 88, Earlswood Road, Belfast, a son.

BENNETT.—On March 8th, 1955, at Okehampton, to Sheila, wife of P. H. G. Bennett, M.R.C.V.S., a son, Jonathan Peter, brother for Karen.

BROOK.—On March 15th, 1955, to Jeanne (*née* Biggers), wife of Alan Brook, MS.R.C.V.S., of 39, Southgate, Chichester, a son, Simon.

FOSTER.—On March 7th, 1955, at Armidale, New South Wales, Australia, to Elizabeth Beatrice (*née* Coward), wife of Alan H. Foster, M.R.C.V.S., a son.

SUTTON.—On January 26th, 1955, at Tunbridge Wells, Kent, to Heather (*née* Streeter) and Peter Sutton, MS.R.C.V.S., a son, a third brother for Janet.

Engagements

WEST—LAURIE.—The engagement is announced between Richard C. A. West, M.R.C.V.S., younger son of Mr. & Mrs. A. J. West, F.C.C.S., of 7, Knollpark Drive, Clarkston, and Mary B. Laurie, younger daughter of Mr. & Mrs. R. Laurie, of 65, Milrig Road, Rutherglen, Lanarkshire.

RUSSELL—ROE.—The engagement is announced between Captain David Gerald Russell, The Royal Inniskilling Fusiliers, elder son of the late Gerald Russell, of Annagassan, Co. Louth, Ireland, and of Mrs. C. M. Richardson, of Glenmore Road, Salisbury, Wiltshire, and Susan Margaret, only child of R. J. Roe, O.B.E., M.R.C.V.S., D.V.S.M., and Mrs. Roe, of 19, Shakespeare Road, Nicosia, Cyprus.

Marriage

MARTIN—BERENT.—At Epsom on March 19th, 1955, Bryan A. Martin, B.V.SC., M.R.C.V.S., of Swindon, to F. Renee Berent, B.A., of Epsom.

R.C.V.S. OBITUARY

PICKERING, Francis, of Long Close, Hexham, Northumberland. Graduated New Edinburgh, May 23rd, 1895. Died March 14th, 1955, aged 83 years.

COMING EVENTS

March

31st (Thurs.). Annual Dinner and Dance of the Yorkshire Division, B.V.A., at the Old Swan Hotel, Harrogate, 7 p.m.

April

1st (Fri.). 9th Annual General Meeting of the A.V.T. & R.W's at Maclay Hall, University of Glasgow, 17, Park Terrace, Glasgow, C.3, 10.30 a.m.

2nd (Sat.). Annual Dinner of the B.R.X. Club, at the Royal Veterinary College, 7 p.m.

7th (Thurs.). Spring Meeting of the North of Scotland Division, B.V.A., at the Northern Hotel, Aberdeen, 2.30 p.m.

15th (Fri.). General Meeting of the Midland Counties Division, B.V.A., at the Star Hotel, Worcester, 2.30 p.m.

20th (Wed.). Meeting of the Royal Society of Medicine, Section of Comparative Medicine, at the Society's house, 5 p.m.

23rd & 24th (Sat. & Sun.). Annual Reunion Week-end for 1952 R.V.C. graduates and contemporaries. Dinner at "Ye Miller of Mansfield" Hotel, Goring, Saturday, 7.30 p.m.

30th (Sat.). Association of State Veterinary Officers Annual General Meeting and Dinner in London.

May

3rd (Tues.). Afternoon Meeting of the Eastern Counties Division, B.V.A., at Colchester.

6th (Fri.). Royal Veterinary College 163rd Annual Ball at the Hyde Park Hotel.

11th (Wed.). Evening Meeting of the Eastern Counties Division, B.V.A., at Ipswich.

12th (Thurs.). R.C.V.S. Dinner, Dorchester Hotel. Further details will be published next week.

31st (Fri.). Annual Dinner-Dance of the Chiron Club in the Royal Hotel, Bristol, 7 p.m.

June

10th (Fri.). Annual Dinner of the R.A.V.C. Officers' Club, at Grosvenor House, Park Lane, London.

September

4th to 10th. 73rd Annual General Meeting and Congress of the British Veterinary Association at Belfast.

APRIL MEETINGS OF COUNCIL AND COMMITTEES, B.V.A., IN EDINBURGH

AT THE ROYAL (DICK) SCHOOL OF VETERINARY STUDIES

Monday, April 4th.—

10.15 a.m. Veterinary State Medicine Committee.
2 p.m. Home Appointments Committee.
4 p.m. Organising Committee.

Tuesday, April 5th.—

10.15 a.m. Parliamentary & Public Relations Committee.
11.30 a.m. Finance Sub-committee.
2 p.m. General Purposes & Finance Committee.

Wednesday, April 6th.—

10.30 a.m. COUNCIL MEETING.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Cumberland. Rectory Farm, Arkleby, Carlisle (Mar. 14).

Dorset. Bucknowle Farm, Corfe Castle, Wareham (Mar. 14).

Kent. Westwood Court Farm, Selling, Faversham (Mar. 17).

Leics. Moorland House Farm, Market Bosworth (Mar.14). Mayfield House Farm, Hose, Melton Mowbray (Mar. 16).

Lincs. Kirkby House, Stickney, Boston (Mar. 15).

Norfolk. Grange Farm, Barney, Dereham (Mar. 12). Broad Farm, Fleggburgh, Gt. Yarmouth. Hole Farm, Hempstead, Holt (Mar. 14).

Northampton. Holme Farm, Yelvertoft (Mar. 14); Top Lodge, Thrapston, Kettering (Mar. 15).

Northumberland. Shotley Field Farm, Shotley Bridge (Mar. 15).

Salop. Ensdon Farm, Montford Bridge, Shrewsbury (Mar. 16).

Staffs. Brick Kiln, Leigh, Stoke-on-Trent (Mar. 15).

Warwicks. Holt Farm, Walton, Rugby (Mar. 14).

Worcs. Lower Inkford, Alcester Road, Wylhall, Birmingham (Mar. 17).

Yorks. Downe Arms, Castleton (Mar. 11); Walnut Farm, Walton, Boston Spa (Mar. 12); Notton Green Farm, Notton, Wakefield; Heygate Farm, Bramham, Boston Spa (Mar. 15); Laurel House, East Barnby, Lythe, Whitby (Mar. 16).

Fowl Pest

Cambs. The Orchard & The Paddock, Jobs Lane, March (Mar. 15).

Ches. Whatcroft Hall, Whatcroft, Northwich (Mar. 12); Hall of the Heath Farm, Haslington, Crewe (Mar. 15).

Norfolk. Rheims House, Three Holes (Mar. 16).

Staffs. Ivy Villa Farm, Wootton, Eccleshall (Mar. 13).

Swine Fever

Ches. Elworth Hall Farm, Elworth, Sandbach (Mar. 16).

Essex. 62, Turner Road, Colchester (Mar. 15); Paynes Farm, Gt. Totham, Maldon; Piggeries, Stubbs Lane, Braintree (Mar. 16); Plains Farm, Ipswich Road, Ardleigh, Colchester (Mar. 17).

Gloucs. Clarks Farm, Little Rissington, Cheltenham (Mar. 15).

Hants. St. Margarets, Fernhill Road, Cove, Farnborough (Mar. 17); Oak Farm, Prospect Road, Cove (Mar. 21).

Kent. Leyton Cross Piggeries, Wilmington (Mar. 18).

Lanarks. Westfield Nursery, Drumchapel, Glasgow (Mar. 21).

Lancs. Glebe Farm, Lunts Heath Road, Widnes; The Piggeries near Taylors Arms, Ford, Liverpool (Mar. 15).

Monmouth. Coombe Farm, Shirenewton, Chepstow (Mar. 21).

Norfolk. Cremorne, Thorpe Road, Thorpe-next-Norwich (Mar. 17); The Beeches, Coltishall Road, Buxton, Norwich (Mar. 21).

Northants. Milton Ham Farm, Milton (Mar. 21).

Renfrew. 6, Market Hill, Castle Semple, Lochwinnock (Mar. 16).

Salop. 2, New Council Houses, Mile Oak, Oswestry (Mar. 15); Weston Coton Farm, Weston, Oswestry; Allotment Cottage, Colemere, Ellesmere (Mar. 16); The Nursery, Bicton, and Hermitage Farm, Hadnall, Shrewsbury (Mar. 18); Llynymapsis Cottage, Morda, Nr. Oswestry (Mar. 19); Sillenhurst Farm, Woore, Crewe (Mar. 21).

Staffs. 25, Best Street, Old Hill (Mar. 16).

Suffolk. Oak Tree Farm, Hepworth (Mar. 18).

Warwicks. 75, Birchy Close, Dickens Heath, Shirley, Birmingham (Mar. 15).

Worcs. 5, Arthur Street, Redditch; "The Cowsheds," Elmley Castle, Pershore (Mar. 21).

Yorks. Hutton Bank Top, Hutton Conyers, Ripon (Mar. 17).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

PREGNANCY TOXAEMIA

Sir,—Mr. J. P. Pickering's comments on the aetiology of pregnancy toxaemia in ewes prompts me to write a few words on this subject. Sheep are so valuable to-day that, in general, I have no difficulty in persuading flock owners to feed well prior to lambing and for some time afterwards.

I think that there is a tendency to lump all deaths together in ewes heavily in lamb and call them pregnancy toxaemia. In spite of supplementary feeding

(hay *ad lib.*, 2 lb. per head per day of equal parts oat and bean meal) losses still occur. Laboratory examination of one such ewe was mainly negative, with a suspicion of mineral deficiency, although ewes were receiving a mineral supplement.

I should be grateful for any advice concerning the feeding of in-lamb ewes as it is most disconcerting that losses should continue when owners are prepared to feed well. If losses are in fact due to pregnancy toxaemia I can only presume that bean protein is inadequate for the ewe's requirements.

Yours faithfully,

J. A. TOOVEY,

Church House,
St. Neots,
Hunts.

March 20th, 1955.

THE DISEASES OF SHEEP

Sir,—We considered the 1949 edition of the N.V.M.A. handbook of sheep diseases to be a well-constructed, useful book of reference, perhaps a little out of date. But how disappointed we are with the 1955 edition prepared by the Technical Development Committee of the British Veterinary Association! We hope that our criticism may stimulate others to give their views and that the result will be an improved edition soon. Basically the book is sound, but it is still out of date.

" In lambs suffering from the streptococcal type of joint-ill sulphanilamide therapy may be exceedingly effective . . ." If sulphanilamide is much cheaper than the more effective sulphonamides there may still be an excuse for using it in large animals: there is no excuse for using it in lambs!

" Antibiotics may be of value." The statement occurs several times. Applied to tick pyaemia as it is on page 35, it does not do justice to Foggie's work on penicillin treatment (Foggie, 1953 & 1954). Practitioners in sheep areas would not treat contagious ophthalmia with " mild antiseptic eye lotions." Chloramphenicol would probably be their first choice and if that failed they might try terramycin or aureomycin. Treatment of contagious pustular dermatitis (orf)—a difficult practical problem—is only lightly touched upon. The authors appear to shrink from imparting information about antibiotics. In general, treatments have not been brought up to date.

Space does not permit a detailed criticism of the book, but we must draw attention to the inadequacy of the paragraph on black disease prevention: there is no recommendation about when or how to vaccinate, and why suggest using *Clostridium oedematiens* anti-serum in the form of human gas gangrene anti-serum when black disease anti-serum is on the market?

The handbook contains some misstatements: strawberry foot-rot is not " an extensive production of granulation tissue extending from the coronet to above the knee or hock . . ." Then the statement that " brucellosis infection of sheep . . . does not occur in the United Kingdom " should surely be modified since Young reported isolation of *Brucella abortus* from an aborted lamb foetus (Young, 1953).

A great deal of work has obviously gone into the preparation of this book. We ask those responsible not to let the matter rest but instead to start revising the book at once with a view to publishing another, better edition.

Yours faithfully,

G. A. M. SHARMAN,
T. GRAHAM MARR,

The North of Scotland College of Agriculture County Advisory Service,
3, Union Street,
Inverness.

March 15th, 1955.

REFERENCES

FOGGIE, A. (1953). *Vet. Rec.* **65.** 169.
———. (1954). *Ibid.* **66.** 690.
YOUNG, S. (1953). *Ibid.* **65.** 247.

METALDEHYDE POISONING

Sir,—I have had occasion to treat this condition in approximately 12 dogs, 30 cats, and three ducks during the past 10 years.

The dogs' symptoms were hypersalivation, hyperaesthesia, inco-ordination or actual convulsions. Except in the more severe cases appetite remained good. The symptoms lasted, with decreasing severity, up to three days. Two cases proved fatal.

Cats' symptoms were similar, with the addition of a marked nystagmus, which I found so consistently as to regard it as of diagnostic significance. The will to eat usually remained but the muscular effort involved was sufficient to precipitate a convulsion, as was also any sudden noise or movement in the animal's surroundings. Recovery was usually complete in 36 hours. Five cases proved fatal.

Ducks showed inco-ordination and torticollis. Spontaneous recovery occurred in two cases, and one proved fatal.

The ducks had not ingested " Meta " directly but had eaten slugs dead or dying from the effects of that preparation.

For treatment I have found nembutal the most useful drug, but in mild cases aspirin has been sufficient to control symptoms. Various injectable calcium preparations and the parenteral administration of magnesium sulphate have apparently produced a beneficial effect in some cases.

Yours faithfully,

OLWEN D. JAMES,

13, Australia Road,
Gabalfa,
Cardiff.

March 21st, 1955.

ERRATUM

Our attention has been drawn to a typographical error in THE RECORD of March 12th. On page 207, col. 1, line 13, the word " expedited " should have read " expected."